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Two Cases of Laryngeal Paralysis, with a Consideration of the Points involved.

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TWO CASES OF LARYNGEAL PARALYSIS,

WITH A CONSIDERATION OF THE POINTS INVOLVED.

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The peruser of recent medical literature, especially in the domain of laryngology, must often be struck with the divergence of views and the discrepancies of statement in works on laryngeal paralysis. There is an undoubted tendency toward bewilderment of mind the more one reads and studies the various experiments to settle the nerve supply and the *modus operandi* of the dilators and closers of the glottis with the complicated interrelation of the various contributory muscles. The laryngologist himself, if his experience is large, must from time to time see certain laryngeal paralyses whose ætiology, after the most exhaustive local and general examination, is a mystery. All other muscular groups are normal, and the patient may otherwise be in good health. Ascribing them to some pre-existing laryngitis or to rheumatism or gout is far from satisfactory without an explanation of how these very common affections, in particular cases, can bring about such results. It has been the lot of the writer to see several such cases when the most careful local inspection and the most painstaking physical examination of the chest failed to disclose

the cause. In most of these cases (all, I think) the immobile vocal cord was in the median line. The chagrin which they have caused has led me to study the two cases presented in this paper with more interest than they would have otherwise excited. They, of course, do not belong to the group of puzzling cases just mentioned :

Case I. *Left Lingual and Laryngeal Paralysis; Atrophy of the Left Side of the Tongue.*—G. K., aged twenty-nine, a German shoemaker, came to the throat department of the Roosevelt Hospital on July 20, 1887. His family history is good. He had typhoid fever when twelve years old; otherwise he has never been sick, though always delicate as a child. He is a moderate drinker of malt liquors. Nine years ago the patient had gonorrhœa and a chancre. He took medicine only four weeks, and had no secondary symptoms nor any trouble attributable to syphilis until four years ago. At that time he began to feel quite weak, but could go on with his work. He states that at that time he was treated for syphilis, but since then has never felt entirely well.

The weak feelings continue and he suffers often from pain in the back of the head and neck, which occasionally extends to the vertex. Last January (1887) he began feeling very much weaker, and noticed that he reeled to the left side when walking in the dark, and felt dizzy. He says his arms and legs felt as though asleep. These symptoms have grown gradually worse. Five weeks ago he suddenly noticed an impediment in his speech. He also began to swallow with difficulty, though he seems undecided as to the time at which this began. He says that when he drinks, the fluids come out of his nose. The patient has a cachectic appearance. His voice is nasal in tone, and he articulates indistinctly. His mental faculties seem normal. There is a slight paresis of the left side of the face, noticeable both in quiescence and when attempting to smile. He can, however, whistle fairly well. His general muscular power seems to be much less than one would expect from his appearance. When he closes his eyes and attempts to walk he falls to the left. On protruding the tongue it is seen to deviate mark-

edly to the left side. There is considerable atrophy of the left side of it, apparent to the eye and appreciated by the finger.* No loss of taste or of tactile sense can be made out. There is no sensory paralysis anywhere. The left palatine arch is completely paralyzed. On laryngoscopic examination, the left vocal cord is seen immovably fixed in the median line. It seems loose and the edge rises slightly with expiration. The right vocal cord overrides it somewhat on phonation.† The cords are both congested. There is abundant muco-purulent secretion on the left side of the naso-pharynx and in the left nostril. There is no sterno-mastoid or trapezius paralysis.

Dr. T. T. Janeway, of this city, kindly made an examination of the patient's vision and of his hearing with the following results:

R. V., $\frac{20}{100}$; with $\frac{1}{36} = \frac{20}{20}$. L. V., $\frac{20}{200}$; with $\frac{1}{36} = \frac{20}{30}$. Ophthalm.: R. $\frac{1}{30}$, L. $\frac{1}{30}$.

H. D. R. W. = 18″. H. D. L. W. = 1″. After inflation, H. D. R. W. = 18″. H. D. L. W. = C.

Aerial sound conduction better in both ears than bone.

Aerial and bone conduction both more reduced in left than in right ear. Tuning-fork in median line heard better in the right ear.

T. F. R., aerial, 19″; bone, 8″. L., aerial, 4″; bone, 3″. After inflation no improvement.

Urinary Examination.—Negative. No sugar. There is no appreciable lung lesion.

Treatment.—The patient was put on large doses of the iodide of potassium. The limit of tolerance seemed to be about half an ounce daily. During intermissions and along with it were given from time to time arsenic and the syrup of the iodide of iron.

The patient was under observation for about a year. Shortly after his first presentation he had three attacks of inspiratory

* The appearance was similar to the case represented in the "Berl. klin. Woch.," No. 29, 1887, reported by Pel.

† I give here the usual appearance. At times I was in doubt as to these minor appearances which are nevertheless so important.

dyspnœa, probably each time from food or drink dropping into the larynx. He also had a quite severe attack from an application of the solution of the chloride of iron to the congested vocal cords with a brush. At the end of a year, when he passed out of observation, there was the following change in his condition:

The dizziness and reeling to the left ceased soon after the commencement of the iodide treatment. The difficulty in swallowing was also soon overcome entirely. His articulation became distinct, though his voice was husky at times. The slight facial paresis disappeared. There was partial though not complete relief from the palate paralysis. His general muscular weakness was relieved so that he could resume work. There was no improvement in the lingual atrophy and paralysis, and no change in the laryngeal condition.

A second examination was made by Dr. Janeway four months after the first, with these results:

Eyes: R. V., $\frac{20}{100}$; with $\frac{1}{36} = \frac{20}{20}$. L. V., $\frac{20}{100}$; with $\frac{1}{36} = \frac{20}{20}$.
Ears: H. D. R. W. 12″. H. D. L. W. $\frac{1}{4}$″.

Aerial conduction better in both than bone.

Bone conduction better in the right than the left.

Tuning-fork gave increase in duration both of bone and aerial conduction, the record being R., aerial, 25″; bone, 10″. L., aerial, 17″; bone, 9″. No improvement after inflation.

Case II. *Left Laryngeal and Sterno-mastoid and Trapezius Paralysis.*—M. M., aged twenty-seven, a married woman and a native of Ireland, came to the Dennet Dispensary April 17, 1888. Her mother died of bronchitis (?); one sister died of consumption.

The patient is an exceedingly nervous and excitable person of limited intelligence. When a child she had lumps in her neck which disappeared later. Four years ago the patient "got a wetting." Since then she has not been unwell and has had "the whites." Alcoholic and specific histories are denied. Two years ago her voice began to be husky at times and she had occasional "choking fits." There was no cough at that time. Six weeks ago the patient began to cough and raise a little blood. She had pain in her back and her leucorrhœa in-

creased. She became short of breath and began to have night-sweats with chilly feelings, followed in the afternoon by fever. Her appetite is poor. She has lost flesh and strength.

Examination.—Her general appearance is fairly good. The circulation in her extremities is poor. Her voice is exceedingly husky. At the apex of the left lung in front there is dullness on percussion, crepitant and subcrepitant râles, increased vocal fremitus, and roughened breathing. There is great tenderness to pressure and percussion over this area, which extends downward to the upper border of the third rib. Posteriorly the signs are not so marked. There is no other pulmonary change. There are no cardiac or aneurysmal indications.

Her voice is unsteady, at times clear, but usually somewhat husky.

The laryngoscope shows the left vocal cord and arytenoid cartilage immovable in the median line. The cord itself is congested and lax, and there is general laryngeal congestion, but no swelling or infiltration. The right cord moves spasmodically. There is no palate or facial paralysis. There is decided paresis of the upper portions of the left trapezius muscle and almost total paralysis of the left sterno-cleido-mastoid, but no restriction of the movements of the head.

It will be noticed in this history that the symptoms referable to the laryngeal paralysis antedated those assignable to the lung lesion about two years. The case when last seen was running the ordinary course of phthisis in the first stages, with no change in the laryngeal symptoms.

Now, in these two cases we have several points of great interest because of the doubts in which they are involved:

1. The peripheral manifestations of a central or nerve lesion.

a. The median position of the affected vocal cords.

b. The concomitant lingual paralysis and atrophy in one case and the paresis of the sterno-mastoid and trapezius muscles in the other, with other symptoms.

2. The character and situation of the lesions, whether in the cerebral cortex, the medulla oblongata, or at some point of the nerve trunks supplying the various muscles.

In order to make the conclusions arrived at clearer, it will be well to review, as briefly as possible, the literature of the subject bearing upon the salient points, and in the order named. Of course, it must be understood that nothing like completeness is intended or aimed at; that would be tiresome and useless.

We need not go back to Galen or even to Albers in prelaryngoscopic days, but all the information of use is to be found in literature since 1860.

It will be noted that the term laryngeal paralysis is applied to these cases. This is for convenience rather than for exactitude of nomenclature. Until recently median position of the vocal cords was generally supposed to be due to paralysis of the crico-arytænoidæi-postici muscles.

As late as 1880 such an authority as Störk (1) said that paralysis of the postici was one of the rarest of laryngeal neuroses. A year later Semon (2) showed conclusively that it was by far the most frequent of all partial laryngeal paralyses. Almost simultaneously with the introduction of the laryngoscope into medicine we find an account of a case of median paralysis of one vocal cord. Lewin (3) gave, in 1860, a wonderfully good description of "paresis of the muscle of the right arytenoid cartilage which narrows the glottis and does not produce hoarseness," in a patient suffering from constitutional syphilis. In the same year Türck (4) described a case of right hemiplegia with immobility of the left vocal cord, which was displaced to the right of the median line. Three years later Gerhardt (5), who is usually credited with reporting the first cases, published his classical work on "Laryngeal Paralysis," with what appears to be the first account of the

laryngoscopic appearances of a case of *double* "posticus paralysis." He reported eighteen cases of laryngeal paralysis in all, and in regard to a cerebral laryngeal center contents himself with saying: "Physiological data do not go far here. The superior roots of the accessorius, like the neighboring vagus, can be traced to the gray mass on the floor of the fourth ventricle." Many cases were afterward reported, and many theories advanced to account for them.

Bäumler (6) and Johnson (7), in 1872 and 1873, and later McCall Anderson (8) and Whipham (9), reported cases of bilateral paralysis of the vocal cords, some of them in the median position, which were due to pressure on one vagus nerve alone. Johnson (10), in another and a very able paper, explained this phenomenon on the strength of the researches of Rosenthal and Waller and Prevost, which were repeated by Professor Rutherford at Johnson's instance. Pressure on the trunk of the vagus may cause bilateral spasm or bilateral palsy, or spasm of one and palsy of the other side of the larynx, by reflex action due to the decussation of the nerve fibers in the medulla, since the vagus is made up of both afferent and efferent nerve fibers, while pressure on the recurrent alone can only cause paralysis of the affected side. This he believed to be the cause of many cases of sudden dyspnœa in thoracic aneurysm. Finally he says: "It is probable that the long-continued irritation of the trunk of the vagus may gradually, as in the cases of traumatic tetanus, induce such demonstrable structural changes in the nerve center as will explain the bilateral palsy, which appears to be one of the results of this chronic nerve irritation." We see here a foreshadowing of part of what Krause years later announced as the results of his investigations. Indeed this article, as pointed out by Semon, has not received the attention and consideration it deserves in the history of laryngeal neurosis. Cohen's (11) recent

remarkable clinical observation of the stimulation of the peripheral sensitive distribution of the pneumogastric in the larynx, causing temporary relief to a flagging respiration, is in line with this early work of Johnson.

Rosenbach (12), in 1880, called attention to the greater frequency of abductor paralysis, and Semon still further emphasized the point, showing that adductor paralysis from a central or nerve lesion was almost unknown.* Since then their respective claims to priority on this point have been urged with a persistence and an industry worthy of a better cause and of a more important subject. Various explanations of this preponderance have been advanced. Rosenbach said that "in the inspiratory closing of the glottis following a paralysis of the dilators there is not primarily a spasm of their antagonists, but a rhythmical perverse innervation in one direction only—namely, toward the adductors." This sentence has been widely quoted, probably on account of the obscurity of its meaning. It is needless to say that it has no experimental proof to stand on, and, if I understand it rightly, can necessarily have none, but must always remain a theory. Semon supposed the adductor and abductor filaments existed separately in the recurrent nerve, and advanced three hypotheses to account for the median position of the cords.

1. The abductor nerve fibers may be peripheral, and hence more exposed to external injury.

2. There may be a specific vulnerability of the abductor filaments, or when adductor fibers are partially disabled the other adductor fibers are able to carry all the adductor stimulus.

* It should be remembered that in *complete* one-sided paralysis of the larynx the vocal cord is in the cadaveric position of Ziemssen, or half way between adduction and abduction.

3. Possibly the nerve fibers receive an increment of nerve force from the superior laryngeal.

McKenzie (13) and Riegel (14) were inclined to accept the first supposition, the former suggesting that the nerve fibers were peripheral, the latter that they were together at one side. Semon himself leaned to the probability of the second hypothesis. It certainly seems to me that claiming a "specific vulnerability" is begging the question and is as unintelligible as Rosenbach's "perverse innervation." It leaves the problem, Why are the abductor filaments singled out for injury? unanswered.

The third hypothesis has received some support from the investigations of Mandelstamm (15) and Exner (16), and in Lennox Browne (17) it has a prominent though not an unreserved supporter. Many years ago Weir Mitchell, as quoted by Johnson, showed that the movements of the glottis in the turtle are due to two sets of muscles, the openers and the closers. The superior laryngeal nerve supplies both, while the inferior supplies only the openers. The superior laryngeal nerves were found to decussate at the base of the brain like the optic chiasm. The superior and inferior laryngeal nerves were separate throughout their course. If all nerves were cut but one superior laryngeal, the larynx movements would still obtain. Schech (18) and Schmidt (19) separately, in 1873, did much to formulate the arrangement of laryngeal innervation and muscular movement given by the text-books and accepted up to a recent date. According to the investigations of Exner and Mandelstamm, the thyreoarytenoid muscle in its internal division receives its innervation almost exclusively from the superior laryngeal through a branch from its pharyngeal division, which they call the middle laryngeal. This muscle is a straightener and tensor of the vocal cord, and is supplied by both nerves on each side equally. The crico-thyreoid muscle is also supplied by

the superior laryngeal nerve of both sides equally, while the recurrent probably furnishes some fibers also.*

"The interarytenoid muscle apparently is supplied equally on the two sides by both the inferior and superior laryngeal nerves. It is probable that the other muscles, including the posticus, receive their principal nerve supply from the inferior laryngeal, though some of it from the superior laryngeal. Often the fibers of the superior laryngeal reach over to the other side posteriorly." Some arrangement of this kind was suspected twenty years ago by Türck (*loc. cit.*, p. 440). It has been urged that this arrangement does not in itself explain those cases where there is a bilateral median position with a central lesion, or a lesion in the vagus above the superior laryngeal. Neither does it explain the cases of Baümler and Johnson, nor why section of the recurrent causes total paralysis.

This brings us to a consideration of the theory of a spasmodic action of all the laryngeal muscles, or at least the principal ones.

Although Jeleneffy states (*loc. cit.*) that he announced in 1872 that the condition was due to spasm from irritation and not to paralysis, and although the same was hinted at by Johnson, as we have seen, the credit of demonstrating and emphasizing the fact by experiments on animals belongs to Krause (21). The conclusion he arrived at as a result of his very careful experiments was, in a few words, as follows: The irritation of pressure or disease, either along

* I have now a case under observation in which there was apparently a paralysis of the thyreo-arytenoid muscles with a paresis of the crico-thyreoid and interarytenoid muscles on both sides. Besides the elliptical opening in the glottis on adduction, the cords had the peculiar rounded outline described by Störk. This latter soon disappeared, but the laxness of the cords still obtains. This condition supervened on the pharyngeal inflammation caused by swallowing carbolic acid. The patient is gradually recovering her voice.

the nerve trunks or in the nerve centers themselves, causes a spasm of all the laryngeal muscles, and the adductor muscles—resembling the flexor muscles of the extremities, to which they have been repeatedly compared—having the greater power, cause the vocal cord of the affected side to assume the median or adducted position. This position may be permanent or it may continue a shorter or longer time, and then, from the progress of the disease and a destruction of the nervous elements, change to one of complete paralysis, when the cords assume the cadaveric position. Of course this is a very crude statement of Krause's very elaborate article, to which reference must be made for the full force of the argument. It may be said here that complete adductor paralysis from a nerve or central lesion is almost or quite unknown, though some good observers have confessed their inability to invariably distinguish between the cadaveric position and complete abduction. Adductor paralysis, usually bilateral, is the rule in the hysterical form, which is never permanent.*

That Krause's conclusions have been subjected to many and very weighty criticisms can not be denied. His opponents, until lately, if not still, are in the majority, but Jeleneffy's work, unless refuted by future investigations, must eventually establish the reality of Krause's conclusions, at least in the main. The former states—and his demonstrations are singularly convincing, at least to the writer—that not only is the crico-arytenoideus posticus an antagonist to

* I have seen one case in which what was without doubt hysterical closure of the glottis was so persistent and caused such extreme inspiratory dyspnœa that tracheotomy with artificial respiration for half an hour were necessary to save the patient's life. Lately, after the lapse of six years, I have seen the patient, who wore the tube for many months. She still has occasional attacks of typical hysterical aphonia with an increase in other hysterical manifestations, but she has had no return of the nearly fatal dyspnœa.

the lateralis in its adducting power, but beyond a certain point is in some of its fibers a synergist. It also tends to fix the arytenoid cartilage on the cricoid, and prevents the vocal process from being depressed by the contraction of the lower fibers of the lateralis. By its counteraction it helps the thyreo-arytenoid and the crico-thyreoid muscles to stretch and straighten the vocal bands. It assists the inter-arytenoideus also to approximate the arytenoid cartilages. The original article* must be consulted for an explanation of this somewhat complicated and yet, according to Jelen-effy's demonstration, very obvious function of the posticus. Krause does not deny the possibility of the occurrence of posticus paralysis, but a little reflection will convince those who have seen many cases of the permanent median position of the cords that it must be a rare one if the above view of the action of the posticus is a correct one. Dr. Solis-Cohen (*loc. cit.*) seems to have adopted Krause's views at least as explaining the cases where there is lesion of the recurrents.

In 1885 Hooper (22) noticed in the dog that there was forcible abduction of the vocal cord from galvanic stimulation of the corresponding recurrent nerve when the animal was very profoundly anæsthetized, and adduction at other times. Krause noticed something of the same phenomenon. The former, therefore, said that the abductors belong to organic life, and denied their proclivity to paralysis. Donaldson (23) in the following year said it was only on weak stimulation that the cords were abducted, and that the abduction was not due to the etherization ; but he also denied the greater proclivity of the abductors to paralysis. Semon and Horsley (24), while denying strenuously the validity of Krause's views, confirmed the statements of Hooper in

* See for *résumé* "N. Y. Med. Journal," Aug. 31, 1889.

regard to deep etherization, as well as those of Donaldson in regard to weak stimulation of the recurrents, but showed that, in repeating Hooper's experiments, Donaldson did not carry the narcosis far enough.

These authors also stated that in excision of the larynx in the living animal the postici muscles lose their contractibility before the other muscles, but Jeleneffy (*loc. cit.*) has shown that this is due to their smaller size and their more exposed condition. While the writer is inclined to accept Krause's views in the main, there are some points which are not explained and some which seem to controvert them. When Remak (25) reported his case of lingual and sternomastoid paralysis with immobility of the right vocal cord, from injury to the nerves at the base of the skull, Fraenkel (26) seemed to think that this settled the question, reasoning that because there was paralysis of other muscles, there could not be spasm of the laryngeal group, but the posticus must be paralyzed. Without at all detracting from the force of this objection, which is open to much more serious criticism, it might be said that the history of laryngeal paralyses abounds in such examples, and the case of Remak is not unique. Case II, recorded here, is such an example. Fraenkel (27) himself has elsewhere advanced an objection that appeared much more vital to the spasm theory. It can not be denied that even in the cases where the lesion is central, and hence including the bulbar origin of the superior laryngeal nerve fibers, the vocal cord, although in the median position, is often, if not usually, lax during respiration, and occasionally is made tense only during phonation. It was difficult to see how this could occur during spasm of all the laryngeal muscles.

Jeleneffy's article explains how it is that after atrophy due to the continued spasm we obtain a condition which is practically a paralysis of the posticus and thus allows a lax-

ness of the cord to occur from the non-fixation of the arytenoid cartilage and the lack of counteraction to the cricothyreoideus and thyreo-arytænoideus internus muscles.

Nevertheless, the objections to the spasm theory are less numerous and weighty than those of paralysis of the posticus alone, since it is supplied by the same nerve as most of the other muscles. This supposition is unsupported by experimental proof, and many well-established facts seem utterly irreconcilable with it. But argument is of little use. Clinical observation can only serve to keep alive the interest in the subject. Physiological experiment and anatomical research, repeated and varied, are the only methods by which the problem will be solved.

The concomitant lingual paralysis and atrophy in Case I is of only contributory interest to us. Although Fairlie Clarke (28), in reporting a case in 1871, declared that he was unable to find any case of lingual atrophy reported in literature, there is at least one by Dupuytren as long ago as 1832 referred to by Leudet (29), while Lande (30) in 1870 reported five cases. Since then, and indeed before, many cases have been reported by the French writers.

The other symptoms need only be referred to in speaking of the location of the lesions. In Case II the shoulder paralysis would indicate, as in Remak's case, that we have to do with a lesion of the vagus and accessory immediately after their exit from the skull. This is still further suggested by the history of glandular enlargement in the neck and by the fact that the phthisis of the left pulmonary apex began two years after the laryngeal affection. Moreover, the "choking fits" or "laryngeal crises" to which the patient had been subject, and the spasmodic action of the right vocal cord, point to an irritation of the vagus, as in the cases of Bäumler and others mentioned. It is reasonable to suppose that there was pressure exerted by some en-

larged cervical gland, although none could be distinguished upon examination.

In Case I, the lesion being evidently an intracranial one, we have two possible locations—a cortical and a bulbar.

The question of a cortical laryngeal center has been the subject of some interesting pathological and physiological investigations. Lewin (31), in 1874, examined a case with left hemiplegia and made out immobility of the left vocal cord in the middle line. There was no autopsy. So scanty are the data upon which to base a reliable localization of a laryngeal center in the cerebral cortex from pathological evidence, that the laryngoscopic appearance of every case of hemiplegia should be recorded for future reference on autopsy. Seguin's (32) cases which were reported in 1877 tended to show that the lower part of the third frontal convolution of the left side, especially in right-handed people, was the seat not only of speech, but of laryngeal movements. This, however, was far from conclusive, as no laryngoscopic examination was made. Indeed, until very recently, either the laryngoscopic or the post-mortem appearance of these supposed cortical lesions has been lacking in each case. Indeed, the case of Garel (33), so far as I know, is the only one in which, with a cortical lesion found at autopsy, there had been in life a laryngeal neurosis noted. There was in his case right hemiplegia with aphasia, and the left vocal cord was paralyzed, thus resembling the case reported by Türck many years ago and referred to above. Here, however, an autopsy was subsequently obtained, and, besides the ordinary hæmorrhagic lesion on the left side of the brain, there was a localized area of softening in the inferior part of the third frontal convolution on the right side. The report is meager in details and no account is given of the microscopic condition of the medulla. Dr. Delavan (34), in a recent paper published in this Journal,

sums up the general testimony on this point in cerebral localization:

"1. Unilateral irritation of a given cortical center excites the corresponding bulbar center and causes bilateral movement.

"2. Unilateral destruction of a given cortical center gives no result, as the influence of the opposite cortical center is sufficient to excite the corresponding bulbar center, and thus to cause bilateral movement.

"3. Bilateral destruction of a given cortical center causes paralysis."

Now, from this standpoint we must conclude that there was a corresponding lesion of the same spot on the opposite side. If so, why was there not bilateral paralysis? A lesion on the left side of the medulla, which, without a microscopic examination, might have been overlooked, would be what we should expect. Garel, however, had a physiological basis on which to stand, and this possibly may have caused him to be a little hasty in his conclusions. Krause (35), in 1884, announced his experiments on the brains of dogs. He demonstrated that in these animals there was a center for the laryngeal adductors in the gyrus præfrontalis, the third frontal or Broca's convolution, near its anterior and descending end. In the neighborhood were also areas for the muscles of deglutition and of the neck. The channel of communication from this region to the medulla is probably through the corpus mamillare. These statements have been confirmed by Semon and Horsley (quoted by Delavan, *loc. cit.*). The electrical stimulation of this cortical area causes different laryngeal muscular actions in different animals—adduction in the monkey and dog, abduction in the cat. Dr. Delavan in the same year (1884) read a paper (36) before the International Congress, at Copenhagen, on a cortical localization of a

laryngeal center, based on a case very much resembling Case I of this paper. This case on autopsy, however, showed the causative lesion to be in the medulla. Gottstein, in the last edition of his work on the throat (p. 302), in referring to the case, pointed out what afterward proved to be the correct situation of the lesion.

There are a sufficient number of carefully reported cases, as well as physiological data, on which to base a diagnosis of and locate a lesion in the medulla oblongata. Hughlings Jackson (37), in reporting some such cases in 1864, which were also seen by Mackenzie (*loc. cit.*), asks the question which now confronts us, "Where is the disease which suddenly produces together paralysis of the tongue, palate, and vocal cord, all on one side?"

His answer was more unsatisfactory than ours should be today, for since then a great deal has been done to supplement the work of Claude Bernard, Lockhart Clarke, and others.

Beginning at the widest part of the floor of the fourth ventricle, where the posterior border of the pons Varolii crosses it, we find an irregular mass of gray matter extending backward and downward to the posterior end of this lozenge-shaped space, where it becomes continuous with the gray matter of the spinal cord. Here, from before backward, the root fibers of the facial, acusticus, glosso-pharyngeal, pneumogastric, and the upper roots of the spinal accessory have their origin. This is not only a motor, but a sensitive tract. The latter, known as the sensitive or ventral nucleus of the vagus, is deeper and a little higher than the dorsal or motor nucleus, though they are continuous with one another. The nucleus of the hypoglossus nerve is nearer the median furrow and parallel with the tract of the other. A careful comparison of Case I, with the symptoms noted and the lesions found in the cases of Senator (38) and Eisenlohr (39) and Delavan (*loc. cit.*), as well as with others, helps us to a

diagnosis and location by pathological evidence. It is probable that the lesion was most extended toward the lower and in the superficial parts of the mass on the floor of the fourth ventricle, where it would involve the nucleus of the hypoglossus and the motor nucleus of the vagus, leaving the sensitive tract uninvaded, since there was no sensory paralysis of any kind noted. It must have extended upward along the median furrow on the left side, involving the nucleus of the acusticus and slightly the facial to account for the facial paresis, the marked diminution of hearing on the left side, and the palate paralysis. The deafness, the difficulty of swallowing, and the tendency to fall to the left we saw improve under treatment. The lingual and laryngeal paralysis remained unchanged, as it usually does. To account for the giddiness, we have either to imagine that it was due to the involvement of the acusticus or to some interference with the blood-supply of the cerebellum. When we come to consider the cause and the nature of the lesion, it is probable, in view of the specific history and the result of treatment, that the patient had areas of softening and congestion due to disease of the coats of the left vertebral artery or its branches, the posterior spinal and the inferior cerebellar. The iodide of potassium relieved the areas where actual tissue necrosis had not taken place, and produced some amelioration of the symptoms, while further improvement could not be expected. It is a singular fact, noticed by Elsberg (40), that, although other muscular groups may recover with varying rapidity and completeness from paralysis, the power of the laryngeal muscles rarely returns. This may be due to the rapidity with which they atrophy on account of their small size.

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